Blood Pressure Management in Neurologic Emergencies

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KEYWORDS

• Blood pressure • Neurologic emergencies • Stroke • TBI • Preeclampsia • PRES

KEY POINTS

- Blood pressure management is crucial in neurologic emergencies to reduce morbidity and mortality.
- Elevated blood pressure in neurologic emergencies can be a modifiable factor with significant prognostic implications.
- Emergency physicians play a key role in managing blood pressure in conditions like stroke, reversible cerebral vasoconstriction syndrome, PRES, traumatic brain injury (TBI), and preeclampsia.
- The review provides a comprehensive framework for managing blood pressure in various neurologic emergencies, including stroke and TBI.
- Current guidelines, emerging evidence, and ongoing controversies in blood pressure management are discussed to improve patient outcomes.

INTRODUCTION AND BACKGROUND

Blood pressure (BP) management is a cornerstone of acute care in neurologic emergencies, where timely and effective intervention is essential to reducing morbidity and mortality. Neurologic complaints constitute about 5% of annual emergency department (ED) visits and are often associated with significant clinical urgency.[1] Elevated BP in these scenarios is not merely a secondary finding but often represents a modifiable factor with significant prognostic implications.[2] Emergency physicians frequently face pressure to act on elevated BP readings, even without signs of end-organ damage.[3] However, this review focuses on scenarios where prompt and precise BP control is essential—such as stroke, traumatic brain injury (TBI), preeclampsia, and other critical conditions—where early recognition and intervention may improve functional outcomes and lower mortality.[4,5] This article aims to provide clinicians with practical insights into current guidelines, emerging evidence, and ongoing controversies, offering a comprehensive, evidence-based resource to enhance patient outcomes in the

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Abbreviations	
AGOG	American College of Obstetrics and Gynecology
AHA	American Heart Association
AIS	acute ischemic stroke
ASA	American Stroke Association
BP	blood pressure
CPP	cerebral perfusion pressure
ED	emergency department
ESO	European stroke
EVT	endovascular therapy
ICH	intracerebral hemorrhage
ICP	intracranial pressure
IV	intravenous
IVT	intravenous thrombolysis
MAP	mean arterial pressure
PRES	posterior reversible encephalopathy syndrome
RCVS	reversible cerebral vasoconstriction syndrome
TBI	traumatic brain injury
TIA	transient ischemic attack
TNK	tenecteplase
tPA	alteplase

complex landscape of neurologic emergencies. This review will begin with a brief overview of hypertension in the ED, followed by a focused discussion on the evidence and BP management strategies for specific neurologic conditions.

Approach to Undifferentiated Hypertension

Hypertension affects nearly 1.3 billion people globally, yet only about 20% have adequate BP control.[6] In the United States, elevated BP is observed in up to 45% of ED visits,[7] but hypertensive emergencies—marked by acute end-organ damage—are diagnosed in only about 0.6% of cases.[6] According to the American College of Emergency Physicians clinical policy,[8] routine testing or initiation of antihypertensive medications for isolated elevated BP without symptoms is not recommended.

The primary role of emergency physicians is to identify hypertensive emergencies through detailed history, vital sign trends, and focused physical examinations assessing cardiovascular, neurologic, ophthalmologic, and renal involvement. Testing should be limited to cases with suspected end-organ damage, using tools such as ECG, metabolic panels, chest radiographs, urinalysis, or brain imaging as needed.

For patients with severe BP elevation but no acute symptoms, management should consider comorbidities, chronicity of BP elevation, follow-up access, and social determinants of health.[9] Prescribing antihypertensive medication at discharge may be reasonable if follow-up is delayed.[10] Ultimately, a patient-centered approach, focusing on distinguishing between transient or chronic BP elevations and true emergencies, helps reduce the long-term burden of uncontrolled hypertension while addressing acute risks appropriately.[11]

Undifferentiated Stroke

Background

The optimal approach to BP management in patients with suspected stroke before advanced imaging remains uncertain, as high-quality evidence guiding prehospital and early emergency care is limited. While extensive research has informed in-hospital BP management for confirmed ischemic and hemorrhagic strokes, evidence for hyperacute BP management in suspected strokes is limited.[12] Elevated BP in

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ischemic stroke may act as a compensatory mechanism to maintain cerebral perfusion in the ischemic penumbra, rapid or excessive BP reduction could theoretically compromise perfusion and exacerbate ischemic injury. In contrast, elevated BP in intracerebral hemorrhage (ICH) can contribute to hematoma expansion, making BP control a potentially beneficial intervention.

For patients eligible for intravenous thrombolysis (IVT), achieving a BP target of $\leq$185/110 mm Hg is critical to minimize the risk of hemorrhagic transformation. This target is based on guidelines from the American Heart Association/American Stroke Association (AHA/ASA) and the European Stroke (ESO).[13,14] However, in the absence of imaging confirmation, the benefits and risks of prehospital BP lowering remain unclear. Effective BP management is an accessible and low-cost intervention, making it a compelling target for improving early stroke outcomes, yet the absence of definitive evidence complicates decision-making in the prehospital and hyperacute settings.

Key trials

- PIL-FAST[15]: A small UK feasibility study testing oral lisinopril in suspected stroke patients with systolic BP greater than 160 mm Hg. The treatment group had lower BP at hospital arrival and 4 hours post admission, but the small sample size (14 patients) and lack of follow-up limited its conclusions.
- RIGHT and RIGHT-2[16,17]: In RIGHT (41 patients), transdermal glyceryl trinitrate (GTN) reduced BP by 18 mm Hg and improved 90-day outcomes. RIGHT-2 (1149 patients) found no functional or mortality benefit, suggesting feasibility but uncertain effectiveness.
- INTERACT-4[18]: This Chinese trial (2404 patients) compared intensive BP lowering (130–140 mm Hg) versus usual care. Outcomes improved in hemorrhagic stroke but worsened in ischemic stroke, underscoring challenges in preimaging BP management.

Management

These trials underscore the complexity of prehospital BP management, with the type of stroke playing a critical role in determining the risks and benefits of early intervention. The European Stroke Organization, reflecting on these findings, currently advises against routine BP lowering in the prehospital setting due to the potential risks associated with misclassifying stroke types.[14]

Lowering BP in suspected stroke before imaging is not currently recommended by international guidelines. However, careful monitoring of BP trends in both prehospital and hospital settings remain an essential component of stroke care. While hypertensive emergencies may warrant BP control upon hospital arrival, this should be balanced with imaging prioritization in accordance with established guidelines. Proactively planning for BP management during imaging and ensuring clear communication with nursing staff and pharmacy, when available, can help facilitate timely and coordinated BP control when necessary.

Conclusion

BP management in suspected stroke before advanced imaging must be individualized based on the patient's clinical presentation and transport context. While achieving a systolic BP less than 185 mm Hg and diastolic BP less than 110 mm Hg is critical for patients being considered for thrombolysis, overly aggressive BP reduction in undifferentiated strokes remains controversial. This balanced approach minimizes potential harm while maximizing the likelihood of favorable outcomes.

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Acute Ischemic Stroke

Background

Acute ischemic stroke (AIS) accounts for approximately 87% of all strokes, with up to 75% of patients presenting with elevated BP on admission.[19,20] While BP elevation may help maintain cerebral perfusion, excessive hypertension is associated with poor outcomes, including hemorrhagic transformation and increased mortality.[12] The approach to BP management varies based on eligibility for IVT or endovascular therapy (EVT), requiring a balance between ischemic risk and hemorrhagic complications.

Blood Pressure Management Based on Reperfusion Eligibility

Nonreperfusion candidates

For AIS patients not receiving IVT or EVT, the optimal BP target remains uncertain. Observational studies suggest a U-shaped relationship, where both very high and very low BP correlate with worse outcomes.[20–22] AHA/ASA and ESO guidelines recommend withholding antihypertensive therapy unless systolic blood pressure (SBP) greater than 220 mm Hg or diastolic blood pressure (DBP) greater than 120 mm Hg, except for specific comorbidities.[13,14] Aggressive BP reduction can impair cerebral perfusion, particularly in large vessel occlusions with impaired autoregulation.

Key trials

- Scandinavian Candesartan Acute Stroke Trial (SCAST)[23]: Randomized 2029 patients with AIS or ICH to candesartan versus placebo within 30 hours. BP reduction was modest, with no effect on functional outcomes, mortality, or recurrence.
- Prevention Regimen for Effectively Avoiding Second Strokes (PRoFESS) trial[24]: Telmisartan versus placebo in 1360 AIS patients within 72 hours. BP differences were minor, with no impact on outcomes.[25,26]
- The Efficacy of Nitric Oxide in Stroke (ENOS) Trial[27]: Transdermal GTN versus placebo in 4011 patients within 48 hours. BP was lower at 24 hours, but there was no sustained functional benefit at 90 days.

Clinical implication: These trials show no consistent benefit from routine BP lowering in AIS patients not undergoing reperfusion therapy. At the bedside, consider BP reduction only for severe hypertension (SBP >220 mm Hg or DBP >120 mm Hg) or for other compelling indications.

Thrombolysis candidates

IVT with alteplase (tPA) or tenecteplase (TNK) is a cornerstone of AIS treatment but increases the risk of ICH, especially with uncontrolled hypertension.[28] AHA/ASA and ESO guidelines recommend lowering BP to $\leq$185/110 mm Hg before IVT and maintaining $\leq$180/105 mm Hg for 24 hours post thrombolysis.[13,14] These recommendations are partly based on the National Institute of Neurological Disorders and Stroke (NINDS) tPA trial that excluded patients with BP greater than 185/110 mm Hg, which was supported by pilot and observational studies.[29–32]

Key trials

- Safe implementation of treatment in stroke (SITS) registry[33]: Observational data found a 4-fold increased risk of symptomatic ICH in patients with SBP greater than 170 mm Hg compared to those within the 141 to 150 mm Hg range.

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- The Enhanced Control of Hypertension and Thrombolysis Stroke Study (ENCHANTED)[34]: Intensive BP lowering (target SBP 130–140 mm Hg) reduced ICH rates but did not improve 90-day functional outcomes, supporting current guidelines of SBP less than 185/110 mm Hg pre IVT and less than 180/105 mm Hg post IVT.

Clinical implication: These findings reinforce strict BP control before and after IVT to minimize hemorrhagic complications. However, aggressive BP lowering beyond guideline thresholds has not demonstrated functional benefit and may risk cerebral hypoperfusion.

Endovascular therapy candidates

BP management for EVT remains an area of active investigation. Elevated BP before, during, and after EVT is associated with an increased risk of hemorrhagic transformation, while excessive BP lowering may impair collateral circulation and infarct salvage.[35] Based on international guideline, BP should be maintained $\leq$180/105 mm Hg during and for 24 hours post EVT, though evidence supporting this threshold is based primarily on early trials where most EVT patients also received IVT.[13,14,36] However, high quality observational data suggests that elevated systolic and/or diastolic BP (>220/120 mm Hg) correlates with worse outcomes, including symptomatic ICH and mortality.[37,38]

Key trial

- BP-TARGET[39]: Compared post-EVT BP targets of 100 to 129 mm Hg versus 130 to 185 mm Hg, finding no significant difference in ICH rates, though adherence to intensive BP goals was low.

Clinical Implication: A BP goal of $\leq$180/105 mm Hg remains reasonable post-EVT, pending further evidence. While extremely elevated BP (>220/120 mm Hg) should be treated, overly aggressive reduction may be harmful.

Pharmacologic management

Intravenous (IV) antihypertensive agents are preferred in acute ischemic stroke due to their rapid onset and titratability. The choice of agent depends on the patient's clinical status, comorbidities, and BP response. Please refer to **Box 1** for reasonable medications with dose ranges.

BP should be monitored every 5 to 15 minutes initially, with adjustments based on patient response. Neurologic checks should be performed frequently to ensure BP reduction is not compromising cerebral perfusion.

Conclusion

BP management in ischemic stroke remains a nuanced clinical decision requiring individualized treatment based on reperfusion eligibility. While the ideal BP target continues to be debated, current evidence supports moderate BP control to prevent hemorrhagic complications while maintaining cerebral perfusion. Ongoing trials will further refine optimal BP thresholds for thrombolysis and endovascular therapy, ultimately improving outcomes in ischemic stroke patients.

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Box 1
Intravenous antihypertensives in neurologic emergencies

Agent	Mechanism of Action	Recommended Use
Nicardipine	CCB	First-line agent; initiate at 5 mg/h IV, titrate by 2.5 mg/h every 5–15 min as needed (max 15 mg/h).[40]
Labetalol	β-blocker with α-blocking effects	Initial bolus: 10–20 mg IV over 1–2 min; may repeat every 10 min with increasing doses (20–80 mg), total cumulative dose not to exceed 300 mg. Alternatively, initiate continuous infusion at 2–10 mg/min following bolus.[40]
Clevidipine	Short-acting CCB	Initiate at 1–2 mg/h IV, titrate by doubling the dose every 2 min as needed (max 21 mg/h).[40]
Hydralazine	Direct vasodilator	Administer 5–10 mg IV over 1–2 min; may repeat as needed (max cumulative dose: 20 mg). Use cautiously due to unpredictable hypotensive response.[40]
Esmolol	Ultra-short-acting β-blocker	Loading dose: 50–500 mcg/kg IV over 1 min, followed by continuous infusion at 25–50 mcg/kg/min. Titrate by 20–50 mcg/kg/min every 5 min as needed (max 300 mcg/kg/min).[40]
Nitroprusside	Arterial and venous vasodilator	Initial: 0.3–0.5 mcg/kg/min; titrate every 5 min to desired effect; usual dose: 3 mcg/kg/min; maximum dose: 10 mcg/kg/min.[40] Avoid unless absolutely necessary (risk of increased ICP).[41]

Intracerebral Hemorrhage

Background

ICH, sometimes referred to as hemorrhagic stroke, accounts for ~10% of all strokes and remains the most lethal subtype, with mortality rates up to 40% despite advances in acute stroke care.[42,43] Elevated BP at presentation is a major modifiable factor, strongly linked to hematoma expansion, neurologic deterioration, and worse functional outcomes.[44,45] The pathophysiology of BP elevation in ICH is multifactorial, driven by sympathetic activation, pain, stress, and chronic hypertension.[46] While anticoagulant use has contributed to rising ICH incidence, the shift to direct oral anticoagulants over vitamin K antagonists may help mitigate this trend.[47,48] Given that most hematoma expansion occurs within the first 3 hours, timely BP reduction is a critical target in acute ICH management.[49] However, clinical trials have yielded neutral or mixed results, leaving uncertainty about optimal BP targets and treatment timing.

Key trials

- INTERACT[50]: In 404 ICH patients, intensive BP lowering (SBP <140 mm Hg) significantly reduced hematoma growth without increasing adverse events
- INTERACT-2[51]: Among 2839 patients, intensive BP reduction showed no difference in death or major disability, but ordinal analysis suggested modest functional benefit. Safety was comparable between groups.
- ATACH-II[52]: Randomized 1000 patients to SBP 110 to 140 mm Hg versus 140 to 180 mm Hg using nicardipine. No functional benefit, but increased renal adverse events with intensive therapy.
- INTERACT-3[4]: An international trial testing a care bundle including BP lowering to less than 140 mm Hg within 1 hour. Functional outcomes improved, but the specific contribution of BP control is unclear due to similar BP levels across groups.

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- INTERACT-4[18]: Prehospital BP reduction (target 130–140 mm Hg) improved outcomes in ICH patients but worsened outcomes in ischemic stroke, highlighting the risk of treating before stroke subtype is confirmed.

Clinical Implication: Early BP reduction to less than 140 mm Hg in ICH is likely safe and may reduce hematoma expansion, though functional outcome benefits are modest. Caution is warranted when treating suspected stroke preimaging, as aggressive BP lowering may harm ischemic stroke patients.

Blood pressure targets and guidelines for intracerebral hemorrhage

Given the mixed trial results, expert guidelines provide practical recommendations:

AHA/ASA 2022 guidelines:[53]

- In patients with spontaneous ICH requiring acute BP lowering, careful titration to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP, can be beneficial for improving functional outcomes.
- In patients with spontaneous ICH in whom acute BP lowering is considered, initiating treatment within 2 hours of ICH onset and reaching target within 1 hour can be beneficial to reduce the risk of hematoma expansion and improve functional outcome.
- In patients with a mild-to-moderate ICH with SBP $\geq$ 150 mg Hg, lowering to 130 to 150 mm Hg is reasonable and safe.
- In patients with spontaneous ICH presenting with large or severe ICH or those requiring surgical decompression, the safety and efficacy of intensive BP lowering are not well established.

ESO 2021 guidelines:[14]

- In patients with acute (<24 hours) ICH, there is continued uncertainty over the benefits and risks (advantages/disadvantages) of intensive blood pressure lowering on functional outcome.
- Hyperacute ICH (<6 hours): Recommend lowering BP less than 140 mm Hg but keeping it greater than 110 mm Hg to minimize hematoma expansion.
- Extended BP Management: Maintain BP control for at least 24 hours and up to 72 hours to reduce ongoing bleeding risk.

Pharmacologic management

IV antihypertensives are preferred due to rapid onset and titratability, ensuring smooth, sustained BP control to avoid large fluctuations, which are associated with worse outcomes.[54] Please see **Box 1** for antihypertensive recommendations.

Conclusion

Blood pressure management in ICH is both time-sensitive and clinically critical. Rapid but controlled BP reduction within the first 2 to 3 hours can limit hematoma expansion, with current evidence supporting a target systolic BP of 130 to 150 mm Hg in most cases. While this strategy appears safe and potentially beneficial, ongoing research aims to further define optimal thresholds and tailor BP management to individual patient profiles.

Transient Ischemic Attack: Management at Discharge

Patients with suspected transient ischemic attack (TIA) who are deemed low risk are often discharged from the ED with neurology follow-up after an initial workup. While the risk of recurrence remains high, it has declined with improved secondary

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prevention.[55,56] RCTs show that prescribing antihypertensives reduces recurrent TIA and stroke risk.[57] Ideally, initiation should be coordinated with outpatient providers, but prescribing at discharge may be appropriate if follow-up delays are expected.

AHA/ASA guidelines and other national recommendations advise continuing, restarting, or initiating antihypertensives after TIA, targeting BP less than 130/80 in most cases.[40,58,59] Thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors, and Angiotensin II Receptor Blockers (ARBs) are first-line options supported by large trials and CCBs are reasonable alternatives if needed.[60,61]

Antihypertensive therapy should be individualized based on comorbidities. Please refer to **Box 2** outlining recommended starting and maximum doses for commonly prescribed agents.

Conclusion

For low-risk TIA patients discharged from the ED, antihypertensive therapy plays a key role in secondary prevention. When timely outpatient follow-up is uncertain, initiating treatment at discharge is reasonable. Targeting a BP less than 130/80 mm Hg is recommended, using agents tailored to comorbid conditions. Thiazide diuretics, ACE inhibitors, and ARBs remain first-line options.

Posterior Reversible Encephalopathy Syndrome

Background

Posterior reversible encephalopathy syndrome (PRES) is a neurologic disorder with acute to subacute onset, presenting with encephalopathy (~80%),headache (~50%), seizures (~75%), visual disturbances (~60%), and focal deficits (5%–15%).[62–65] First described in 1996 as reversible posterior leukoencephalopathy syndrome, it is characterized by vasogenic edema, typically in the parieto-occipital regions on MRI.[64,66,67] Though hypertension is a common feature (~70%), it is absent in up to 20% of cases.[62] PRES remains understudied, with most data derived from case series and meta-analyses.[63]

PRES affects all ages but is most common in women aged 30 to 50 years.[66,67] It is associated with hypertension, renal disease, sepsis, immunosuppressive therapy, autoimmune disorders, and preeclampsia.[63,67–70] Two theories explain its pathophysiology: the hyperperfusion theory, which attributes it to BP fluctuations causing vascular leakage[62,63]; and the toxic theory, which links endothelial dysfunction from endogenous or exogenous toxins, particularly in immunosuppressed and septic patients.[71]

Box 2
Standard oral antihypertensives

Agent	Mechanism of Action	Starting Dose/Max Dose	Considerations
Hydrochlorothiazide	Diuretic	12.5–25 mg once daily/50 mg daily[40]	First-line for patients without comorbidities
Lisinopril	ACE Inhibitor (ACEi)	5–10 mg once daily/40 mg daily[40]	Preferred for patients with diabetes and proteinuria
Losartan	ARB	25–50 mg once daily/100 mg daily[40]	Suitable for patients intolerant to ACE inhibitors

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Pharmacologic management

BP control is the cornerstone of treatment, though no trials have assessed its direct impact on outcomes.[69,72] Consensus amongst experts reducing SBP by ≤25% in the first hour, with normalization over 24 to 48 hours, while avoiding excessive BP fluctuations.[63] First-line antihypertensives include clevidipine, labetalol, and nicardipine (**Box 1**). Early identification and management of underlying triggers, such as infection, medications, or autoimmune disorders, are essential.[69,73] Worrisome medications include, but not limited to, immunosuppressive agents, chemotherapy agents, monoclonal antibodies, bone marrow stimulating agents, and high dose steroids.[73] Seizures should be treated with anticonvulsants, but prophylaxis is not recommended.[69] Magnesium should be maintained within a normal range due to its neuroprotective effects.[69] Given the risk of status epilepticus and intracranial hemorrhage as well as frequent use of IV antihypertensives, intensive care unit (ICU) monitoring is often warranted.

Conclusion

PRES requires early recognition, BP control, and management of underlying triggers. Careful hemodynamic monitoring and targeted therapy can improve outcomes and prevent complications.

Preeclampsia and Eclampsia

Background

Hypertensive disorders of pregnancy are among the leading cause of maternal and perinatal mortality worldwide, responsible for approximately 16% of maternal deaths.[74] Preeclampsia is defined as new-onset hypertension (systolic BP ≥140 mm Hg or diastolic BP ≥90 mm Hg) after 20 weeks gestation accompanied by proteinuria or signs of end-organ dysfunction.[74] Eclampsia refers to the onset of seizures in women with preeclampsia. These conditions present after 20 weeks gestation, during labor, or within 6 weeks postpartum, though onset before 34 weeks is uncommon.[75]

Emergency physicians should promptly evaluate BP ≥140/90 mm Hg in pregnant or postpartum patients and urgently treat severe hypertension (≥160/110 mm Hg) to prevent complications such as stroke, heart failure, and myocardial ischemia.[74] Preeclampsia is diagnosed by sustained hypertension plus proteinuria or end-organ dysfunction (severe features).[74] Risk factors include antiphospholipid syndrome, assisted reproduction, obesity (body mass index [BMI] >30), chronic hypertension, diabetes, kidney disease, age ≥35, multifetal gestation, obstructive sleep apnea, lupus, prior preeclampsia, and thrombophilia.[76] Though examination findings may be minimal, new-onset edema, petechiae, or right upper quadrant (RUQ) tenderness warrant further evaluation.[74] Please refer to **Box 3** for preeclampsia diagnostic criteria, modified from the American College of Obstetrics and Gynecology (AGOG) practice guideline.[74]

Pharmacologic management

Emergent antihypertensive therapy is indicated for severe preeclampsia (SBP ≥160 mm Hg or DBP ≥110 mm Hg, or with severe features), ideally within 30 minutes of confirmation.[74] For preeclampsia without severe features, BP management should be promptly discussed with obstetrics. Magnesium sulfate administration is essential for seizure prophylaxis in severe preeclampsia.

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Box 3
Diagnostic criteria for preeclampsia (*Asterisk indicates severe features*)

	Criteria
BP	• SBP ≥140 mm Hg or DBP ≥90 mm Hg on 2 occasions, ≥4 h apart after 20 wk gestation in a patient with previously normal BP
	OR
	• SBP ≥160 mm Hg or DBP ≥110 mm Hg (can confirm within minutes to expedite treatment)*
AND	Proteinuria
	• ≥300 mg per 24-h urine collection
	• Protein/creatinine ratio ≥0.3 mg/dL
	• Dipstick reading of 2+ (only if other methods unavailable)
OR*	In the absence of proteinuria, new-onset hypertension with the new onset of any of the following:
	• Thrombocytopenia: Platelet < 100,000/μL
	• Renal insufficiency: Serum creatinine >1.1 mg/dL or doubling from baseline (without other renal disease)
	• Hepatic Dysfunction: Liver transaminases ≥2 times normal
	• Pulmonary edema
	• Neurologic symptoms: Persistent headache unresponsive to medication (excluding alternative diagnoses), visual disturbances

Please refer to **Box 4** which are recommended antihypertensive regimens, in accordance with AGOG guidelines with no significant efficacy differences among choices.[74]

Conclusion

Effective management of preeclampsia and eclampsia in the ED emphasizes prompt diagnosis, controlled BP reduction, seizure prophylaxis, obstetrics consultation, and vigilant monitoring to improve maternal and fetal outcomes.

Box 4
Antihypertensive regimens in preeclampsia/eclampsia

Agent	Dose	Notes
Labetalol	10–20 mg IV initially; then 20–80 mg every 10–30 min (max 300 mg) or continuous infusion 1–2 mg/min IV.	Avoid with asthma, cardiac dysfunction, or bradycardia.
Hydralazine	5–10 mg IV every 20–40 min (max cumulative dose 20 mg), or infusion 0.5–10 mg/hr	May cause maternal hypotension or abnormal fetal heart tracings.
Nifedipine (immediate release)	10–20 mg orally; repeat in 20 min if needed, then every 2–6 h; max daily dose 180 mg	May cause reflex tachycardia and headaches

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Reversible Cerebral Vasoconstriction Syndrome

Background

Reversible cerebral vasoconstriction syndrome (RCVS) is a rare, underrecognized cause of thunderclap headache, marked by transient cerebral artery narrowing.[77] It can cause stroke, TIA, hemorrhage, and PRES due to impaired autoregulation.[78] Triggers include serotonergic drugs, vasoconstrictors, stimulants (eg, cocaine, and methamphetamines), cannabis, postpartum state, and physical or emotional stress.[78] RCVS predominantly affects women (2–10:1 ratio), with onset around age 45.[79] Angiography often reveals a classic *sausage-on-a-string* pattern.[80] BP management is important in RCVS, as both hypotension and hypertension may worsen outcomes.[77] About 25% to 30% of patients experience BP surges during their course.[81] While no standard BP target exists, keeping SBP less than 160 mm Hg appears reasonable, given the higher hemorrhagic risk above this threshold.

Pharmacologic management

CCBs like nimodipine (30–60 mg q4h) and extended-release verapamil (120 mg q12–24h) are used to relieve vasospasm in RCVS.[77] However, they have not shown consistent benefit in outcomes and may increase ischemic risk if BP drops too low.[81,82] A reasonable approach is to titrate BP based on symptoms—aiming to reduce thunderclap headache severity while avoiding hypotension—and maintain SBP below 160 mm Hg to minimize the risk of hemorrhage without compromising cerebral perfusion.

Conclusion

RCVS is a complex cerebrovascular condition characterized by reversible arterial narrowing and significant blood pressure fluctuations, requiring careful management. While calcium channel blockers are commonly used, individualized blood pressure control remains critical to minimize complications and improve outcomes. Further research is needed to establish optimal treatment strategies.

Traumatic Brain Injury

Background

Traumatic brain injury (TBI) is a major source of morbidity, mortality, and disability, affecting an estimated 2.9 million people annually in the United States with over 60,000 deaths and 5 million living with long-term deficits.[83] TBI severity ranges from concussion to coma. Blood pressure management is critical, as both hypotension and hypertension worsen outcomes.[84]

Following TBI, cerebral autoregulation is impaired, making cerebral blood flow highly dependent on maintaining an optimal cerebral perfusion pressure (CPP), which is determined by mean arterial pressure (MAP) minus intracranial pressure (ICP).[85] Hypotension reduces cerebral perfusion pressure (CPP = MAP – ICP), risking ischemia, hypoxia, and neuronal death—strongly linked to increased mortality.[86] Conversely, hypertension can raise ICP and cerebral edema, also worsening prognosis.[87]

Optimal blood pressure targets

The 2017 Brain Trauma Foundation guidelines recommend maintaining SBP greater than 100 mm Hg for patients aged 50 to 6, and greater than 110 mm Hg for those 15 to 49 or over 70, though evidence was insufficient for strong endorsement.[84] Recent studies suggest no single threshold, but rather a BP range associated with reduced mortality.[88] The 2023 3rd Edition Prehospital Guidelines emphasize avoiding

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hypotension and recognizing that both low and high BP can worsen outcomes, a principle extendable to ED care.[89]

Key studies

- Chesnut RM, and colleagues:[90] A prospectively collected data set from the Traumatic Coma Data Bank demonstrated that a single episode of hypotension was associated with a 2-fold increase in mortality.
- EPIC TBI study (secondary analysis)[88]: In over 12,000 prehospital TBI cases, mortality rose sharply with SBP less than 90 mm Hg, but risk increased across the 40 to 119 mm Hg range. The lowest adjusted mortality was observed with SBP 130 to 180 mm Hg.
- IMPACT study[91]: A retrospective cohort examining the relationship of thresholds for SBP and MAP demonstrated SBP ranges from 120 to 150 mm Hg and MAP ranges from 85 to 110 mm Hg are thresholds to target improved outcomes.

Clinical implication: Maintaining SBP ≥110 to 120 mm Hg, and ideally in the 130 to 150 mm Hg range, is associated with better outcomes in TBI. Hypotension—even brief or mild—can double mortality, and high-quality evidence supports shifting prehospital and in-hospital targets upward from the traditional 90 mm Hg threshold. Frequent BP monitoring and proactive correction are essential in minimizing secondary brain injury.

Pharmacologic management

Volume resuscitation is essential in trauma care to maintain perfusion and oxygen delivery to the brain. Hypotension should be corrected promptly with isotonic fluids or blood products.[89] Hypertonic fluids, such as hypertonic saline or mannitol, may be used in severe TBI (Glascow Coma Scale [GCS] <8) when elevated ICP is suspected.[89] No fluid type has shown superiority in outcomes, and albumin is contraindicated due to increased mortality.[92] Vasopressors like norepinephrine and phenylephrine are used to maintain CPP, though data comparing agents are limited.[84] Outcomes appear similar between agents, so selection should be guided by clinical context (eg, sepsis and neurogenic shock).[93]

Conclusion

BP management in TBI is a delicate balance—both hypotension and hypertension can worsen brain injury and negatively affect outcomes. Guidelines recommend maintaining SBP greater than 100 mm Hg for patients aged 50 to 69 and greater than 110 mm Hg for others, with emerging evidence supporting a target range of SBP 110 to 150 mm Hg. Preventing and promptly correcting hypotension while avoiding excessive hypertension is crucial to preserve cerebral perfusion and reduce secondary brain injury. Tailored resuscitation strategies using fluids and vasopressors should be guided by patient-specific factors. Further research is needed to refine precise BP targets and optimize treatment strategies in this complex population.

SUMMARY

Optimal BP management in neurologic emergencies requires a nuanced, patient-centered approach, balancing the risk of cerebral hypoperfusion against complications associated with excessive hypertension. While current evidence and guidelines inform BP targets across various neurologic conditions—including ischemic stroke, intracerebral hemorrhage, preeclampsia/eclampsia, PRES, RCVS, and traumatic brain injury—significant controversies and research gaps persist. Emergency

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clinicians must carefully integrate clinical presentation, underlying pathophysiology, evolving literature, and expert recommendations to individualize BP management strategies. Future randomized controlled trials will be essential to refine these guidelines further, enhancing patient outcomes through precise, evidence-based interventions tailored to each neurologic emergency.

CLINICS CARE POINTS

- Differentiate hypertensive emergencies from incidental hypertension: treat only when there is acute end-organ damage or compelling indication; isolated ED hypertension does not require immediate reduction.
- Stroke care hinges on timing and subtype: in acute ischemic stroke, maintain BP $\leq$185/110 mmHg for thrombolysis/EVT; in intracerebral hemorrhage, early smooth lowering to 130–150 mmHg reduces hematoma expansion.
- Avoid overcorrection: aggressive or rapid BP reduction can worsen cerebral ischemia (AIS, RCVS, PRES) or cause renal injury (ICH); target gradual, controlled lowering.
- Pregnancy-related hypertension is unique: severe preeclampsia/eclampsia ($\geq$160/110 mmHg) requires treatment within 30 minutes with labetalol, hydralazine, or nifedipine, plus magnesium for seizure prophylaxis.
- Traumatic brain injury demands prevention of hypotension: maintain SBP >100–110 mmHg depending on age; both hypotension and severe hypertension are linked to increased mortality.

DECLARATION OF AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the author(s) used ChatGPT (OpenAI) and Copilot (Microsoft) in order to assist with manuscript editing, summarizing evidence, and refining clarity of content. After using these tools/services, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication. This declaration does not apply to the use of basic tools for checking grammar, spelling, references, etc.

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